

# Swimming Pool Injuries, Mycobacteria, and Tuberculosis-Like Disease

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**A**BRASIVE accidents in swimming pools are not rare. They may occur in diving, in getting in or out of the pool, or in underwater swimming. Most frequently such accidents involve the bridge of the nose, the elbows, or the knees. Although in themselves they are seldom of consequence, it has recently been recognized that this type of accident may lead to inoculation lupus vulgaris, granulomatous tuberculosis lesions, or other tuberculosis-like lesions.

## **Mycobacterium tuberculosis**

Hellerström (1), in 1951, reviewed six cases of inoculation lupus vulgaris, some of which he had reported as early as 1939 (2). They were all associated with swimming-pool injuries. Describing the clinical features, which were strikingly uniform, he wrote:

Within an area of a couple of square centimeters or more on the bridge of the nose an eruption developed, which consisted of soft papules, reddish-violet to reddish-brown in color, and ranging in size from a pinhead to a split pea; some of the papules were topped by crusts and coalesced. Two of the cases presented elevated ulcers measuring 15 by 15 and 7 by 7 millimeters, respectively. In the major proportion of cases the initial abrasion had healed when the papules appeared in the vicinity. On diascopic examination the papules showed the distinctive color of lupus nodules, and they were easily penetrated by a blunt probe applied with slight pressure. Hence, the clinical diagnosis was lupus vulgaris. The regional lymph nodes were either not at all, or only slightly to moderately, enlarged.

In one case Hellerström observed acid-fast bacilli in the lesions, thus affording some laboratory confirmation to his diagnosis.

Later Hellerström (3), using more sensitive laboratory techniques, was able to isolate the tubercle bacillus from one swimming pool. He

felt that the problem of skin tuberculosis acquired in swimming pools was complex and far from being solved. He suggested that, although tuberculosis control officers and sanitary engineers have an interest, "tuberculosis as a waterborne infection is a problem that calls for the attention of dermatologists . . ."

Also in 1951, Cleveland (4) reported four cases from Canada. He concluded that "the clinical and histopathologic appearance of the lesions was strongly suggestive of tuberculosis cutis" although "no acid-fast bacilli were demonstrated in the lesions . . ." All Cleveland's cases were associated with the same swimming pool, which was filled with tidal sea water and was under good sanitary control. He believed that the pool contamination might have resulted from the discharge of urine or sputum from an infected person. In summary, Cleveland suggested that "tuberculous infection may occur more often than the absence of reported cases would indicate."

In commenting on these papers, Sulzberger and Baer (5a) suggested that the etiology was questionable. They pointed out that the smegma bacillus may be present in pools in large numbers and also that this organism when inoculated into the skin produced a "tuberculoid type of response."

## **Mycobacterium balnei**

In a comprehensive monograph, Linell and Nordén (6) summarized previous studies and described an epidemic of 80 cases of benign skin ulceration in Örebro, Sweden. They isolated a previously unknown *Mycobacterium* very similar to Koch's bacillus and gave it the specific name *balnei*.

Linell and Nordén's cases were characterized by a papular lesion of spongy consistency which was typically located on the outside of the elbow. The lesion grew slowly and eventually crusted, with scaling of the surrounding skin. Thick secretion developed under the crust and healing proceeded slowly, leaving a bluish-red

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soft scar. The whole sequence could last 2 years, especially if complicated by purulent infection. Histologically most of the lesions were granulomatous, and acid-fast bacilli were demonstrated in only one case. However, the organism was isolated from both the water and the walls of the swimming pool associated with the epidemic, and laboratory studies on animals and human volunteers (the authors) proved conclusively that *M. balnei* was the etiological agent. No new cases occurred after the pool was rebuilt, replacing the rough concrete walls with smooth tiles, and the chlorination system made more effective.

Zettergren, cited by Linell and Nordén (6), reported a similar episode of 60 cases in Västcrås, Sweden. Although *Mycobacterium marinum* was considered the etiological agent by Zettergren, Linell and Nordén isolated *M. balnei* from lesions and from the pool. The term "mycobacteriosis balnearea" was applied to the syndrome. When breakpoint chlorination was introduced, the epidemic was brought completely under control.

#### Other Reported Cases

Brück (7), in 1951, described 3 cases of inoculation tuberculosis, 2 of which were associated with swimming pool injuries. In 1952, he reported another case of inoculation lupus (8). Isolation of the tubercle bacillus from the lesions completely confirmed the clinical diagnosis. These findings led Brück to conclude that "so-called swimmer's lupus," or mycobacteriosis balnearea, could be separated into two types: (a) cases in which *M. tuberculosis* was the causative agent, and (b) cases in which *M. balnei* was responsible for the infection. He placed his 1952 case and those of Hellerström in the first category, and the cases of Linell and Nordén and Zettergren in the second. Hellerström, in a paper already cited (3), reviewed all of the available case histories and concluded with Brück that similar clinical manifestations may have been due to different etiological agents. He suggested, however, that the organism isolated by Linell and Nordén might be a mutant of *M. tuberculosis*, or that it might be *M. marinum* as believed by Zettergren. He was unwilling to accept *M. balnei* as the responsible agent.

Tolmach and Frank (9), in the United States, reported another case of inoculation lupus vulgaris, which was characterized as skin granuloma with tubercle formation of unknown etiology. This infection resulted from a nose abrasion in a swimming-pool accident.

Nine cases of post-swimming-pool abrasion infections diagnosed as tuberculosis verrucosa cutis were observed by Rees and Bennett (10) in San Francisco. The clinical picture was quite different from that given by Hellerström. As to the etiology, Rees and Bennett ruled out granuloma due to silicates and deep fungus infections, but they were unable positively to define the agent. They mentioned the possibility of infection by the smegma bacillus or by *M. tuberculosis*, although neither was demonstrated in the lesions or the pool.

In the 1954-1955 Year Book of Dermatology and Syphilology (5b), Sulzberger and Baer reviewed the monograph by Linell and Nordén and commented as follows: "This is a masterly clarification of a relatively new entity apparently caused by a quite newly discovered acid-fast mycobacterium [*M. balnei*]. It appears virtually certain that this is the entity previously described by Hellerström in Sweden, D. E. H. Cleveland in Canada, Rees and Bennett and Jesse Tolmach and S. B. Frank . . . in the United States. It is small wonder that this infection of the bridge of the nose and other sites was in the past often considered a type of tuberculosis, and it is likely that the uninitiated will continue in this error in many future cases."

#### Conclusion

It is clear from the foregoing review that a new disease entity—a lupuslike dermatitis—and possibly a new means of transmitting skin tuberculosis are now known. What is not known is how widespread or frequent the infections are. The finding of cases in Europe, Canada, and the United States would indicate widespread geographic distribution. Those concerned with the operation of swimming pools should be aware of the potential hazard from this source, and the clinician should consider swimming-pool trauma in the diagnosis of tuberculosis-like skin infections.

From the limited data available, it would

appear that swimming-pool construction and sanitation may play a significant role in controlling the spread of this disease entity. Smooth-surface walls and breakpoint chlorination have proved effective in curtailing epidemics.

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